LAPAROTOMY

The abdomen was opened through a paramedian incision alongside the previous scar. On reaching the peritoneum it was found only in the uppermost third of the incision. The lower two-thirds was extraperitoneal and occupied by a grossly distended bladder, which was drawn up towards the umbilicus. The peritoneal cavity was opened and the incision extended upwards. The lower segment showed pronounced oedema and bruising. The uterovesical peritoneum was opened, bringing into direct view a fetal limb through a concomitant rupture of the previous caesarean scar and the urinary bladder. The entire fetal head was located within the bladder, which stretched over it like a bathing cap. The remainder of the fetus was in the uterine cavity.

The ruptured transverse scar was widened under vision with a pair of scissors and the baby delivered as far as the neck by breech extraction. The head, which was in the occipitoposterior position, was then extracted from the bladder. This resulted in the birth of a healthy male infant with an Apgar score of 6/10 at one minute. All layers of the bladder were involved in a laceration from the trigone for a distance of about 8 cm measured in the partly contracted state after delivery. In addition to the ruptured scar a vertical laceration extended from it to beyond the cervix into the vagina for a few centimetres. The edges of the ruptured bladder were firmly adhered to the lower segment, from which it could be separated only by sharp dissection. The ureteric orifices were visible on each edge of the laceration.

Both ureters were catheterized. The bladder was dissected free with some difficulty because of its adherence. Total hysterectomy was performed with conservation of the Fallopian tubes and ovaries. Suprapubic cystotomy was carried out with a Malécot catheter. The bladder was repaired in two layers and extraperitonealized, the ureteric catheters being removed at the end of the operation. The vaginal vault was incompletely closed to allow drainage. One litre of blood was transfused and a course of ampicillin prescribed. Except for pyrexia on the first and second days the postoperative period was uneventful. The suprapubic catheter was removed on the ninth day and both mother and baby were discharged on the 14th day. Four weeks later the mother was found to be asymptomatic and the baby thriving. An intravenous pyelogram showed no abnormality.

Comment

Extensive laceration of the lower uterine segment may be expected to result in laceration of the adjacent urinary bladder because of the close anatomical relation of the two organs. This is remarkably uncommon, and in a series of uterine ruptures reported by one of us (Hassim, 1967) only 13% showed some form of laceration of the bladder. Oedema of the bladder, bruising, and varying degrees of haematoma occur more often.

If the fetus is not wholly or partly retained in the uterus, as happens in most uterine ruptures, it may be expelled into the peritoneal cavity or rarely into the broad ligament. In a case of pelvic outlet contraction the fetus was expelled and completely retained in the vagina (Morgan, 1960), while in another case extrusion into the bladder had occurred (Devi, 1962). Devi postulated that extrusion of the fetus into the bladder is possible only if the lower segment and bladder are adherent to each other so that rupture of both organs occur simultaneously.

In the present case a haemorrhagic lower segment due to placenta praevia at the last delivery may have necessitated many sutures before haemostasis was secured. This would encourage healing by fibrosis and possible adherence to the bladder wall. The actual rupture through the bladder must have occured while the patient was being transferred to the operating theatre, because vaginal examination had previously confirmed the presence of the presenting part in the vagina. Early diagnosis and prompt measures resulted in survival of the infant.

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Diabetes Insipidus with an ACTH-secreting Carcinoma of the Bronchus

C. DAVIDSON

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Polyuria commonly occurs in patients with the ectopic adrenocorticotrophic hormone (ACTH)-secreting syndrome (Allott and Skelton, 1960) and is usually ascribed either to glycosuria or to the effect of hypokalaemia on the kidneys. Alternatively hypothalamic metastases may produce true diabetes insipidus. In the following case there was evidence of both defective secretion of antidiuretic hormone (ADH) and an impaired renal response to administered ADH. An unusual feature was the correction of the renal defect by a large dose of dexamethasone.

Case Report

The patient, a 46-year-old labourer, was first seen at the chest unit in 1968 with pulmonary tuberculosis, which was successfully treated with para-aminosalicyclic acid and isoniazid up to his present admission. In 1969, however, routine chest *x*-ray examination showed a hilar shadow which at bronchoscopy proved to be an inoperable oat cell carcinoma. At that time there were no obvious metastases and he was treated with radiotherapy (2,500 rads).

In January 1970 his condition deteriorated over a few weeks and he was admitted to hospital with a vague history of weight loss, thirst, and polyuria. He was wasted, confused, and dehydrated and was noted to have pronounced pigmentation of Addisonian distribution. There was generalized lymphadenopathy suggesting carcinomatous spread but no radiological evidence of recurrence in the chest. He had 2% glycosuria without ketones and the blood sugar was 350 mg/100 ml. Plasma electrolytes after rehydration showed a hypokalaemic alkalosis—Na^+ 144, K^+ 2·5, Cl^- 89, HCO_3^- 36 mEq/l., urea 24 mg/700 ml—and this with the other findings suggested the ectopic ACTH-secreting syndrome. This was supported by high plasma immunoreactive ACTH and fluorogenic corticosteroid levels which did not suppress with dexamethasone 8 mg daily for three days (Table I). Liver function tests and serum calcium and magnesium levels were within normal limits. He was initially treated with spironolactone 400 mg/day and potassium chloride 128 mEq/day, and after 10 days the plasma potassium and

TABLE I—*Plasma Levels of Cortisol and ACTH before and after Dexamethasone Administration*

	On Admission	After Dexamethasone
Plasma cortisol in µg/100 ml (midnight : 9 a.m.)	46 : 46	45 : 47·8
Plasma ACTH in pg/ml (midnight : 9 a.m.)	1,200 : 720	1,300 : —

Whittington Hospital, London N.19
C. DAVIDSON, M.R.C.P., Senior House Officer (Present address: St. Bartholomew's Hospital, London E.C.1)

bicarbonate levels returned to the normal range and remained so until his death six weeks later.

Despite improvement of the electrolyte disturbance and control of the glycosuria by small doses of insulin the patient continued to have pronounced polydipsia and polyuria. The urine output was 6-8 litres/24 hours, with urinary osmolarities between 60 and 200 mOsm/l. There was no evidence of renal tubular damage (no casts, proteinuria, or aminoaciduria) but the creatinine clearance was 54 ml/min. The response to vasopressin tannate (10 units in oil intramuscularly) was a maximum urinary osmolarity of only 360 mOsm/l. occurring 12 hours after injection. This partial renal response to vasopressin is evident from Fig. 1, which shows that

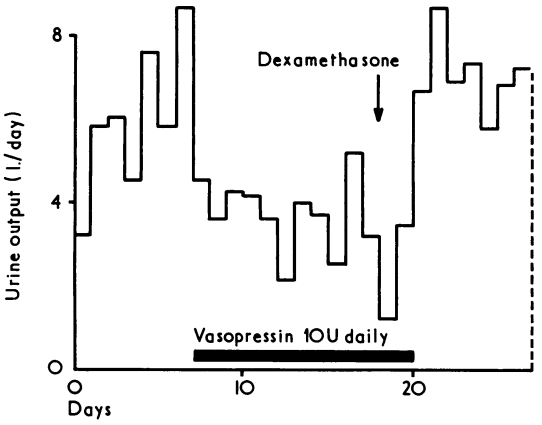

FIG. 1—Effect of vasopressin tannate (10 units daily intramuscularly) on urinary output. Dexamethasone was given in a dose of 8 mg on day 18 only.

the urine output was maintained at from 2 to 4 litres/24 hours by daily intramuscular injections of vasopressin tannate in oil 10 units. After 10 days the renal response to vasopressin was little better than before (maximum urinary osmolarity 404 mOsm/l.) and he remained confused. Although there were no localizing neurological signs he subsequently developed generalized and then focal epileptic attacks suggesting intracerebral metastases. The polyuria was therefore thought to be the result of both hypothalmic metastases and the observed defect in renal concentration.

The renal defect showed a further unusual feature, which was found by coincidence during a dexamethasone suppression test. Dexamethasone administered at the same time as vasopressin restored a normal renal response, as shown in hourly urine collections over consecutive days (Table II).

TABLE II—*Renal Response to Vasopressin and Dexamethasone*

	Range of Urinary Osmolarity (mOsm/l.)	Urine Volume (litres/24 hr)
Vasopressin 10 units intramuscularly	100–403	4·0
Dexamethasone 10 mg intramuscularly	55–215	5·4
Vasopressin + dexamethasone ..	103–695	1·3

This proved to be reproducible in the chronic as well as the acute situation and the effect is seen in Fig. 2; dexamethasone given with vasopressin restored urine osmolarities to the normal range (500-750 mOsm/l., as established by Miles, *et al.* (1954)). The urine output fell to 500-1,500 ml/24 hours and the severe thirst abated for the first time since the patient's admission. It is also appropriate that on the two occasions when vasopressin was stopped while dexamethasone was continued urine output rose towards its former level, indicating little change in the underlying defect. The initial dose of dexamethasone was 8 mg/day. The effect, however, appeared to be maintained when this was reduced to 1·5 mg/day, but the patient died before this could be examined further. The duration of his illness had been about three months.

At necropsy there was general dissemination of tumour in the lymph nodes, liver, and lung. The adrenals were almost entirely replaced by large metastases and there were widespread deposits throughout the brain, with involvement of both the posterior

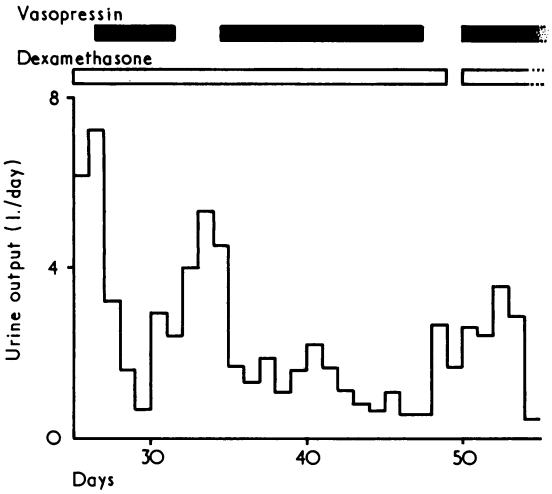

FIG. 2—Effect of oral dexamethasone (8 mg/day) on urinary output, given with and without vasopressin (10 units daily intramuscularly).

pituitary and the inferior part of the hypothalamus. The kidneys were macroscopically and microscopically normal.

Comment

In contrast to the classical Cushing's syndrome hypokalaemia is the usual finding in patients with the ectopic ACTH-secreting syndrome (Liddle *et al.*, 1969) and is thought to reflect the severity of corticosteriod secretion (Prunty *et al.*, 1963). Although serum potassium levels in this case were restored to normal by therapy and there was no objective evidence of intracellular potassium depletion the abnormal response to ADH remained unchanged. This response is characteristic of the hypokalaemic kidney (Rubini, 1961) and it seems possible that a cellular potassium deficiency remained in spite of normal serum levels.

The unexpected effect of dexamethasone on urine output in the presence of already high levels of corticosteroids is not readily accounted for. This effect was observed only in the presence of exogenous ADH, suggesting a synergistic action on the kidney. There was no evidence to indicate that the underlying metabolic abnormality had been altered, since plasma ACTH levels were not suppressed. Corticosteroids are necessary for normal renal tubular function, and in their absence medullary osmolarity is impaired —a finding also present in potassium depletion. Cellular potassium depletion has been observed to influence cell membrane activity (Essig and Leaf, 1963), and this may partly underly the functional abnormality observed in the renal tubule in potassium depletion, particularly the failure to respond to ADH. Corticosteroids are known to stabilize cell membranes (Fell and Weiss, 1965), and it is possible that in these large doses dexamethasone restored the normal cellular response to ADH by this mechanism.

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